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TECHNICAL REPORT

Methodology Report for Phosgene Model (CGModel)

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14. ABSTRACT

Phosgene (NATO designation CG) was used as a chemical weapon in WW I. When inhaled it acts on lung tissues (and eyes) by hydrolysis and acylation reactions, causing irritation and damaging of pulmonary tissue membranes leading to respiratory failure and eventually cardiac failure and death, at high enough exposures. Given a vapor exposure, CGModel calculates four time values related to injury progression. Using a time dependent severity vector that quantifies the physiological effects of the exposure, the model then associates severity vectors with phosgene exposure and then maps each severity vector to a performance value. The performance function can then calculate the time to recovery and return to duty, or else time to death as the two outcome probabilities.

15. SUBJECT TERMS

Phosgene; human performance; casualty estimates; health effects

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CONVERSION TABLE

Conversion Factors for U.S. Customary to metric (SI) units of measurement.

MULTIPLY → BY → TO GET

TO GET ← BY ← DIVIDE

angstrom	1.000 000 x E -10	meters (m)
atmosphere (normal)	1.013 25 x E +2	kilo pascal (kPa)
bar	1.000 000 x E +2	kilo pascal (kPa)
barn	1.000 000 x E -28	meter^2 (m^2)
British thermal unit (thermochemical)	1.054 350 x E +3	joule (J)
calorie (thermochemical)	4.184 000	joule (J)
cal (thermochemical/cm^2)	4.184 000 x E -2	mega joule/m^2 (MJ/m^2)
curie	3.700 000 x E +1	*giga bacquerel (GBq)
degree (angle)	1.745 329 x E -2	radian (rad)
degree Fahrenheit	$t_k = (t°f + 459.67)/1.8$	degree kelvin (K)
electron volt	1.602 19 x E -19	joule (J)
erg	1.000 000 x E -7	joule (J)
erg/second	1.000 000 x E -7	watt (W)
foot	3.048 000 x E -1	meter (m)
foot-pound-force	1.355 818	joule (J)
gallon (U.S. liquid)	3.785 412 x E -3	meter^3 (m^3)
inch	2.540 000 x E -2	meter (m)
jerk	1.000 000 x E +9	joule (J)
joule/kilogram (J/kg) radiation dose absorbed	1.000 000	Gray (Gy)
kilotons	4.183	terajoules
kip (1000 lbf)	4.448 222 x E +3	newton (N)
kip/inch^2 (ksi)	6.894 757 x E +3	kilo pascal (kPa)
ktap	1.000 000 x E +2	newton-second/m^2 ($N-s/m^2$)
micron	1.000 000 x E -6	meter (m)
mil	2.540 000 x E -5	meter (m)
mile (international)	1.609 344 x E +3	meter (m)
ounce	2.834 952 x E -2	kilogram (kg)
pound-force (lbs avoirdupois)	4.448 222	newton (N)
pound-force inch	1.129 848 x E -1	newton-meter (N-m)
pound-force/inch	1.751 268 x E +2	newton/meter (N/m)
pound-force/foot^2	4.788 026 x E -2	kilo pascal (kPa)
pound-force/inch^2 (psi)	6.894 757	kilo pascal (kPa)
pound-mass (lbm avoirdupois)	4.535 924 x E -1	kilogram (kg)
pound-mass-foot^2 (moment of inertia)	4.214 011 x E -2	kilogram-meter^2 ($kg-m^2$)
pound-mass/foot^3	1.601 846 x E +1	kilogram-meter^3 (kg/m^3)
rad (radiation absorbed dose)	1.000 000 x E -2	**Gray (Gy)
roentgen	2.579 760 x E -4	coulomb/kilogram (C/kg)
shake	1.000 000 x E -8	second (s)
slug	1.459 390 x E +1	kilogram (kg)
torr (mm Hg, 0^0 C)	1.333 22 x E -1	kilo pascal (kPa)

*The Becquerel (Bq) is the SI unit of radioactivity; 1 Bq = 1 event/s.

**The Gray (Gy) is the SI unit of absorbed dose.

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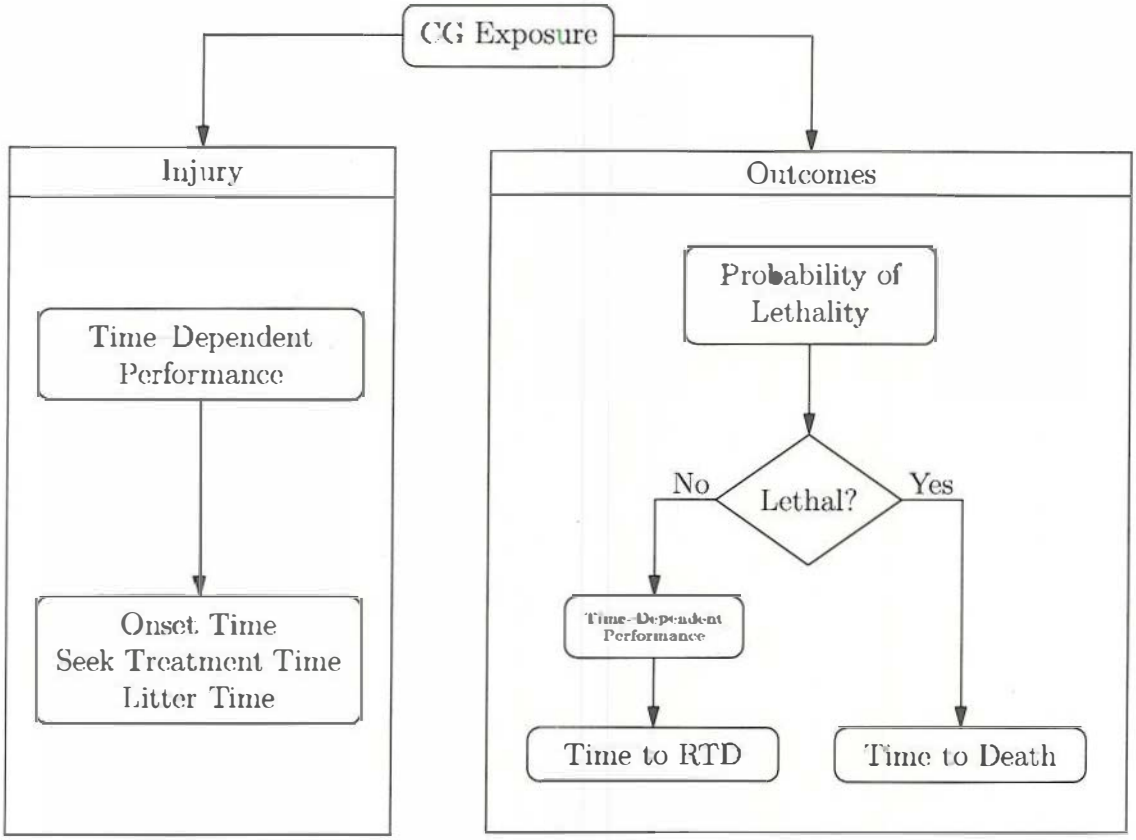

Figure 1: Overview of CGModel calculations.

The Java class CGModel models the human health effects of exposure to phosgene (CG) gas, including human performance capability during and after exposure. CGModel's methodology for estimating performance decrement is well established [2, 3, 6, 12–14] and underlies the casualty estimates generated by modules such as HDModel, GBModel, VXModel, and ClModel. In this report we describe this methodology and show how we implemented it in CGModel.

Figure 1 illustrates the key values calculated by CGModel. Given a CG vapor exposure, CGModel estimates four time values related to the progression of injury: onset time, seek treatment time, litter time, and either time to return to duty (RTD) or time to death.

Figure 2 illustrates the two main steps in CGModel's performance calculation. First, CGModel estimates the severity of the signs and symptoms of injury resulting from a phosgene exposure. This estimate, a time-dependent severity vector, represents a quantification of the physiological effects of the exposure. The four components of the severity vector, real numbers between 1 and 5, correspond to four sign/symptom (S/S) categories and describe the severity of the effects associated with each category. In Section 1 we list the S/S categories used by CGModel, give descriptions of the severity values 1, 2, 3, 4, and 5 (for each S/S category), and describe how CGModel a sociates severity vectors with phosgene exposures. Second, CGModel maps each severity vector to a performance value P, where

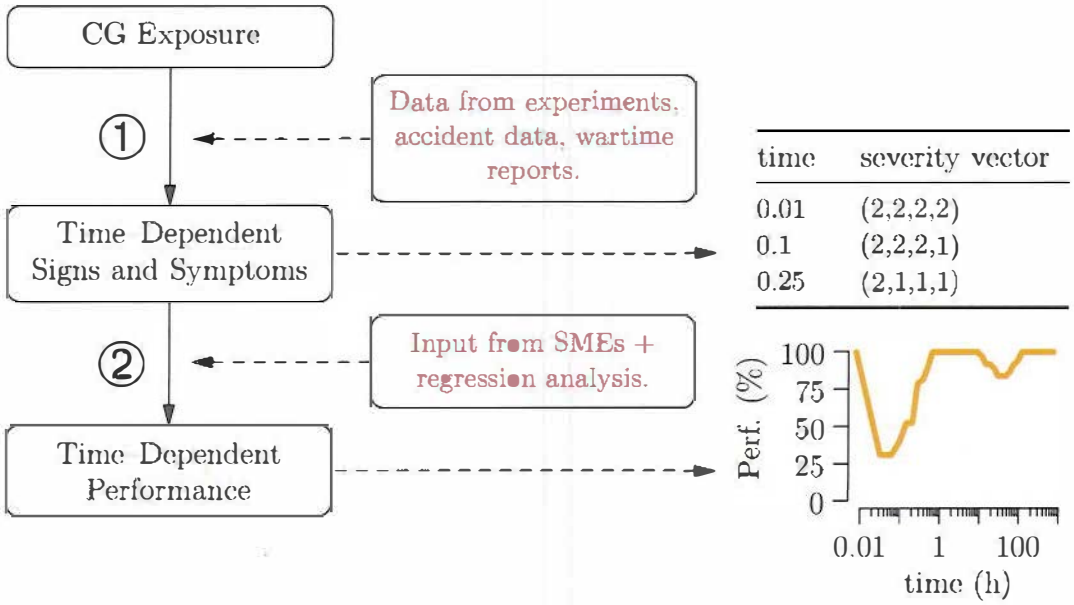

Figure 2: Overview of CGModel performance calculation. Step ① utilizes data from laboratory experiments, data from accidental releases of phosgene, and wartime reports. Step ② utilizes SME (Subject Matter Expert)-provided performance decrement estimates and regression. Lower right graph shows performance curves associated with increasingly severe exposures.

$0 \leq P \leq 1$. This task–specific mapping models the increase in task time due to phosgene exposure. In Section 2 we give the performance function P and plot performance curves $(100 \cdot P(t))$ for several exposure scenarios. In Section 3 we show how CGModel uses $P(t)$ to estimate time phased casualties. Finally, in Section 4 we describe how CGModel estimates time to RTD or time to death.

1 Signs and Symptoms

In this section we describe how CGModel associates a time dependent severity vector with a given phosgene exposure (i.e., Step ① in Figure 2). Before giving details about Step ①, however, we must briefly discuss Step ②. Two key steps in defining Step ② are the following:

1. determine the performance decrements associated with a base set of severity vectors;
2. derive the performance model's regression coefficients (see Equation 2), so that performance predictions can be made for arbitrary severity vectors.

In previous applications of this methodology, the first task mentioned above was accomplished by interviewing SMEs or conducting performance assessment batteries. For this study, however, time constraints necessitated an alternative approach. Therefore, we defined Step ② by utilizing regression coefficients from a previous human response model [14]. That

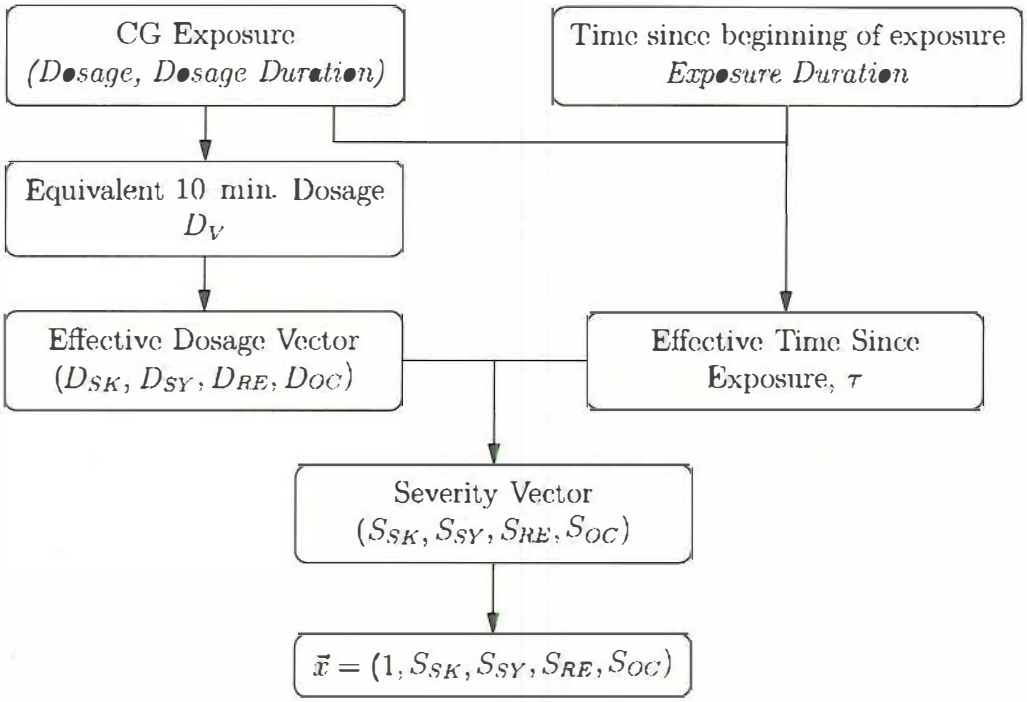

Figure 3: Calculation of the severity vector and the sign/symptom (S/S) complex $\vec{x}$ from a given phosgene exposure and the elapsed time since the beginning of the exposure (corresponds to Step ① in Figure 2).

model, which describes the effects of exposure to mustard (HD), utilized the S/S categories and severity levels given in Table 1. Therefore, CGModel also uses Table 1 to describe symptom severity. In summary, the symptomatology associated with phosgene exposure (which can be regarded as a subset of the symptomatology associated with HD exposure) can be adequately described using Table 1, so we used Table 1 and the regression coefficients from a previous study [14] as the basis for Step ②.

The S/S categories and severity level descriptions given in Table 1 therefore form the basis of mapping ①, which is outlined in Figure 3. First, the HE/RC Manager passes *Dosage*, *Dosage Duration*, and *Exposure Duration* to CGModel. *Dosage*, the time integral of concentration, and *Dosage Duration*, a measure of the effective duration of the exposure, define the phosgene exposure; *Exposure Duration* refers to the elapsed time since the beginning of the exposure. From these three parameters CGModel then calculates three intermediate values: the equivalent 10 minute dosage D_V, the effective dosage vector $(D_{SK}, D_{SY}, D_{RE}, D_{OC})$, and the effective time since exposure τ. Finally, CGModel calculates the severity vector $(S_{SK}, S_{SY}, S_{RE}, S_{OC})$. In the paragraphs below we describe each of these calculations.

Equivalent 10–Minute Dosage CGModel's symptom severity table (Table 7) associates severities with 10 minute dosages, so CGModel estimates the equivalent 10 minute vapor dosage D_V from the supplied *Dosage* and *Dosage Duration* values. CGModel calculates

Table 1: CG S/S categories and descriptions of severity levels. In a previous modeling effort [14], these signs and symptoms were used to describe the effects of HD exposure.

Skin Damage		
	1	No effect.
	2	Skin sensitive to touch in crotch, armpits, and on inside of elbow and knee joints.
	3	Skin sore in crotch, armpits, elbow and knee joints, and painful when moving, red swollen skin, tiny blisters on hands and neck.
	4	Skin raw and painful in crotch, armpits, elbow and knee joints, red swollen body skin, large blisters on hands and neck.
	5	Skin peels off leaving open raw areas and painful blisters.
Systemic Damage		
	1	No effect.
	2	Nauseated and swallows often.
	3	Headache, nauseated, vomited once or twice.
	4	Headache and fever, vomited several times and will again.
	5	Pounding headache, dry heaves, fatigued from vomiting.
Respiratory Damage		
	1	No effect.
	2	Dry mouth, dry cough, sneezing, and runny nose.
	3	Sore throat, continuous coughing, hoarse voice, chest feels tight.
	4	Hurts to breathe, hacking cough, cannot speak.
	5	Awful chest pain, wheezing and short of breath, coughs up red colored mucus.
Ocular Damage		
	1	No effect.
	2	Eyes sting and tear.
	3	Eyes feel gritty and sensitive to light, nonstop tears flood eyes.
	4	Eyelids puffy and eyes burn, painful to keep open.
	5	Eyelids swollen shut and burning eyes too painful to open.

Table 2: Acute Exposure Guideline Level (AEGL) values for phosgene (mg/m^3) (AEGL-1 values are not defined). [18, p.18]

Classification	10 min	30 min	1 h	4 h	8 h
AEGL-1	NA	NA	NA	NA	NA
AEGL-2	2.5	2.5	1.2	0.33	0.16
AEGL-3	15	6.2	3.1	0.82	0.34

Table 3: 1 Hour Air MEG (military exposure guideline) for phosgene (mg/m^3) [24, p.C 27],[25, p.C-2-16]

Health Effect Level		
Minimal	Significant	Severe
0.4	1.2	3.0

D_V by assuming that certain combinations of exposure time and phosgene concentration cause the same time dependent symptomatology (see Figure 4) and applying an interpolation/extrapolation algorithm. We estimated the shape of the isoeffect loci in Figure 4 by considering exposure standards and guidelines (see Tables 2 and 3). To associate physiological effects with a given contour (i.e., to develop Table 7, which describes time dependent symptomatology), however, we considered data from multiple sources in the literature.[1]

Effective Dosage Vector After calculating D_V, CGModel calculates effective dosages for each S/S category (see Table 4). Note that CGModel does not model liquid exposures and that CGModel assumes protection is unavailable. CGModel uses the effective respiratory dosage D_{RE} to calculate patient condition codes (see Table 6).

Physical exertion can significantly intensify the effects of phosgene and cause phosgene's effects to appear earlier [23, p.II 3], [26]. CGModel describes this potentiation using the factor F, which depends on D_V, the respiratory minute volume, and the level of physical activity (see Tables 4 and 5). Currently, CGModel uses regression coefficients for the 105 mm artillery loader (see Sec. 2), so the factor F is set to model heavy activity.[2] The effective

[1] We note that, because we based our isoeffect calculations on AEGL and MEG values, it would be more accurate to say that each contour represents exposures that are "similarly disabling." Thus, our model assumes that "similarly disabling" implies "similar symptomatology." This is not necessarily true, of course, and a more detailed model would be needed to describe certain characteristics of S/S severity due to phosgene exposure. For example, "exposures to 2 ppm for 80 minutes will not cause any irritation but result in pulmonary oedema some 12 - 16 hours later" [20, p.150]. This exposure implies $D_V = 516$ mg·min/m^3 (see Table 23). However, if $D_{OC}(= D_V) = 516$, our model predicts eye irritation immediately after the exposure (see Table 7).

[2] If the task (upon which the performance calculation is based) can be changed (by users of the CGModel

Level 1	Level 2	Level 3
(10, 0.4)	(10, 2.5)	(10, 15)
(30, 0.4)	(30, 2.5)	(30, 6.2)
(60, 0.4)	(60, 1.2)	(60, 3.1)
(240, 0.2)	(240, 0.33)	(240, 0.82)
(480, 0.1)	(480, 0.16)	(480, 0.34)

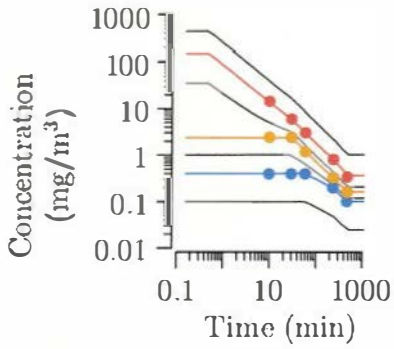

Figure 4: Exposures ((exposure time, concentration)) used to estimate the equivalent 10 minute vapor dosage D_V. Exposures in a given column are assumed to cause the same time dependent symptomatology. Time unit is minutes, concentration unit is mg/m^3. The data points on which these curves are based illustrate that Haber's Law is valid for phosgene "within certain limits" [8, p.A-2].

Table 4: Effective dosage calculations in CGModel (F represents potentiation due to physical exertion, M_V represents respiratory minute volume)

S/S Category	Effective Dosage
Skin	$D_{SK} = D_V$
Systemic	$D_{SY} = D_V \cdot \frac{M_V}{M_V^{\text{Normal}}} \cdot F$
Respiratory	$D_{RE} = D_V \cdot \frac{M_V}{M_V^{\text{Normal}}} \cdot F$
Ocular	$D_{OC} = D_V$

systemic dosage D_{SY} and the effective respiratory dosage D_{RE} depend on F; therefore, F affects both performance and the probability of lethality (see Eq. 3). In effect, F reduces the median lethal dosage (by 10% for light activity, 25% for heavy activity).

Effective Time Since Exposure The symptom severity vector also depends on the effective time since exposure τ, where

$$\tau = \max\left\{0, ExposureDuration - \left(\frac{DosageDuration}{2}\right)\right\} \quad (1)$$

class) in future versions of this software, the activity level upon which F is based could be made adjustable, too.

Table 5: Definition of potentiation factor $F(D)$, where $D = D_V \cdot \frac{M_V}{M_V^{\text{Normal}}}$ is measured in mg·min/m^3 (potentiation is not modeled for D less than 100 mg·min/m^3; see Table 26).

Light Activity	Heavy Activity
$F = \begin{cases} 1, & D < 100 \\ 1 + \frac{D-100}{8.1\text{LCt}_{50}-900}, & D \geq 100 \end{cases}$	$F = \begin{cases} 1, & D < 100 \\ 1 + \frac{D-100}{2.25\text{LCt}_{50}-300}, & D \geq 100 \end{cases}$

Table 6: Patient condition code calculation in CGModel. Code = 0398: pulmonary agent with early (< 4 hours) symptoms, Code = 0399: Pulmonary agent with delayed (> 4 hours) symptoms. See Table 12.

Effective Respiratory Dosage D_{RE} $\left(\frac{\text{mg·min}}{\text{m}^3}\right)$	Code
$D_{RE} < 182$	399
$182 \leq D_{RE}$	398

Figure 6 illustrates the relationships among *Exposure Duration*, *Dosage Duration*, and the effective time since exposure τ.

Severity Vector and S/S Complex CGModel calculates the severity vector $(S_{SK}, S_{SY}, S_{RE}, S_{OC})$ using Table 7 and an interpolation algorithm. Finally, the S/S complex $\vec{x}$ is defined by

$$\vec{x} = (1, S_{SK}, S_{SY}, S_{RE}, S_{OC}).$$

The value of the first component of the S/S complex, 1, results from the regression analysis that underlies the performance function in Equation 2.

2 Performance

Mapping ② in Fig. 2 was constructed using SME input and logistic regression, as described in section 8.3 of [14]. Specifically, CGModel utilizes the following performance function to map S/S complexes $\vec{x}$ to performance values P ($0 \leq P \leq 1$):

$$P = \frac{1}{1 + \exp\left(-\vec{x} \cdot \vec{\beta}\right)} \tag{2}$$

Table 7: Ⓐ. Severity values for the four dosage bands used by CGModel. The number 0 represents 1111 ("no effect" for all S/S categories). Exposure range refers to effective 10–minute dosages D_{SK}, D_{SY}, D_{RE}, and D_{OC} (not the equivalent 10–minute vapor dosage D_V; see Table 4). Ⓑ. Effective times since exposure for severity values in corresponding column of Ⓐ. In this table (i.e., before CGModel applies its smoothing algorithm), for $\tau < 0.01$ hours or $\tau \geq 336$ hours, $(S_{SK}, S_{SY}, S_{RE}, S_{OC}) = (1,1,1,1)$. This table is based on data from experiments, wartime reports, and data from accidental releases of phosgene (see Sec. 6). Note: These four columns actually define the severity functions for the four specific effective dosage values G_i, $1 \leq i \leq 4$ (i.e., not for entire dosage ranges; see Fig. 7).

Exposure Range (Effective 10–min. Dosage, mg·min/m^3	4.1 – 81	81 – 182	182 – 1215	1215 – 2025
S/S Severities	0	2222	2333	2343
S_{SK}, S_{SY}, S_{RE}, S_{OC}	1121	2221	2332	2222
	0	2111	2111	2111
	0	0	2111	2131
	0	1121	0	1131
	0	1131	1121	1241
Ⓐ	0	1121	1131	1351
	0	0	1241	1241
	0	0	1131	1131
	0	0	1121	1121
	0	0	0	0
Effective Time Since	0.01	0.01	0.01	0.01
Exposure τ (hours)	24	0.1	0.1	0.25
	48	0.25	0.25	0.5
		0.5	0.5	2
		12	2	3
		24	4	4
Ⓑ		60	8	6
		96	12	96
			72	168
			120	240
			168	336

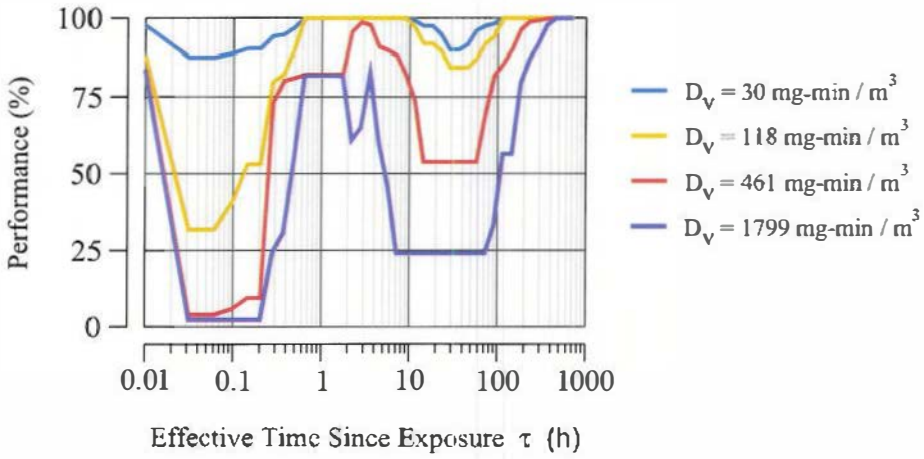

Figure 5: Performance curves ($100 \cdot P(t)$) for several values of D_V (respiratory minute volume = 15 L/min).

(this is Equation 3.3 in [14]). For the reasons discussed at the beginning of Sec. 1, CGModel uses the same task-specific regression coefficient vectors $\vec{\beta}$ as are used in HDModel.[3] Moreover, to ensure that the performance value approaches 1 as dosage approaches 0 (that is, as the symptom severities all approach 1), CGModel incorporates the stretching algorithm described in Section 8.3 of [14]. Fig. 5 illustrates $100 \cdot P(t)$ for several values of D_V (assuming respiratory minute volume = 15 L/min).

3 Onset Time, Seek Treatment Time, Litter Time, Work Efficiency

CGModel uses $P(t)$ to calculate onset time (τ_{onset}), seek treatment time (τ_{seek}), and litter time (τ_{litter}). The onset time equals the amount of time that elapses between the point at which $ExposureDuration = DosageDuration/2$ and the point at which $P = 0.995$. The seek treatment time and litter time are calculated in a similar manner (see Table 8 and Figure 6).

CGModel calculates work efficiency according to Tables 9 and 10 (see Sec. 4 for the definition of τ_{RTD}).

4 Outcomes: Time to RTD, Time to Death

Time to Death CGModel estimates the probability of lethality P_L to be

$$P_L = \Phi\left(PS \log_{10} \frac{D_{RE}}{LCt_{50}}\right) \tag{3}$$

[3] Currently, CGModel uses $\vec{\beta} = (6.647001, -1.425135, -0.786148, -0.625109, -0.900659)$, the regression coefficients for the 105 mm artillery loader [14, pp.24-25].

Table 8: Performance thresholds.

Time	Performance Threshold
Onset (τ_{onset})	0.995
Seek Treatment (τ_{seek})	0.75
Litter (τ_{litter})	0.25

Table 9: Calculation of work efficiency for the case in which $\min\{P\} \leq 0.75$.

Case	Work Efficiency
$\tau < \tau_{onset}$	100
$\tau_{onset} \leq \tau < \tau_{seek}$	$100 \cdot P$
$\tau_{seek} \leq \tau < \tau_{RTD}$	0
$\tau \geq \tau_{RTD}$	80

Table 10: Calculation of work efficiency for the case in which $\min\{P\} > 0.75$.

Case	Work Efficiency
$\tau < \tau_{onset}$	100
$\tau_{onset} \leq \tau$	$100 \cdot P$

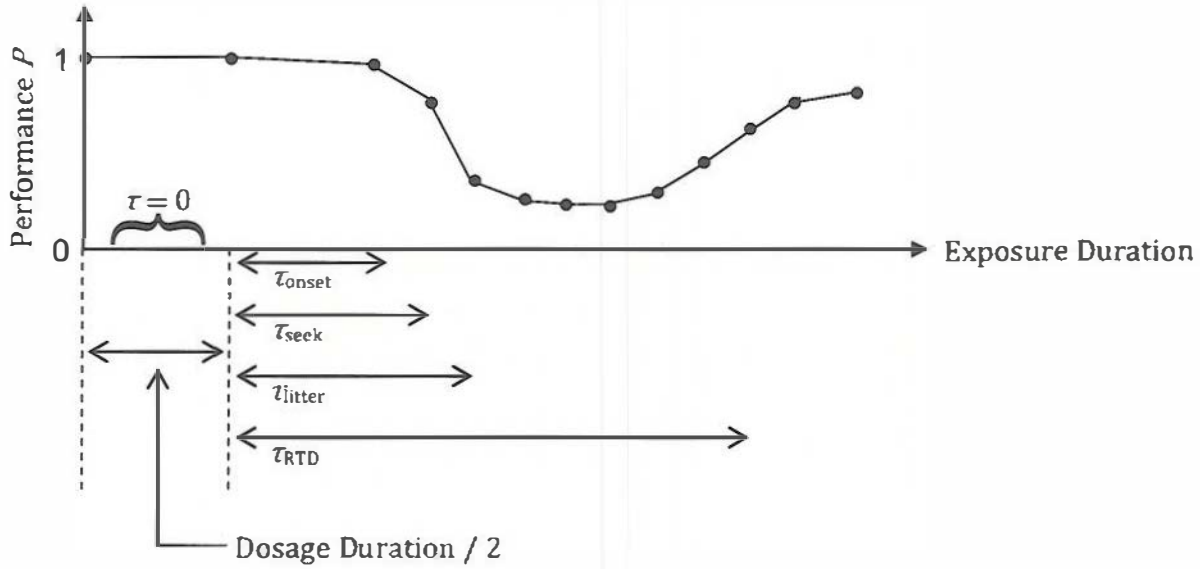

Figure 6: Notional performance vs. effective time since exposure τ and exposure duration.

where D_{RE} is the respiratory system effective dosage (see Table 4), PS is the probit slope, LCt_{50} is the median lethal dosage, and Φ is the cumulative distribution function of the standard normal distribution (model parameters are given in Table 11). CGModel then determines the lethality of the exposure based on a random number $Ran \in [0, 1]$ that is passed to CGModel. If $Ran \leq P_L$, the exposure is considered lethal and the time to death is set equal to 1.5 days (see Table 32). If $Ran > P_L$, the exposure is nonlethal and CGModel calculates τ_{RTD}.

Time to RTD If the exposure is nonlethal and τ_{seek} is defined (i.e., minimum performance is less than or equal to 0.75), CGModel uses $P(t)$ to calculate τ_{RTD}, the time to RTD. If τ^* equals the time that elapses between the point at which $ExposureDuration = DosageDuration/2$ and the point at which P returns to 0.75 (see Fig. 6),

$$\tau_{RTD} = \max\{\tau^*, \tau_{seek} + 24 \text{ hours}\}. \tag{4}$$

Time to RTD is not defined if the minimum performance is greater than 0.75.

5 Assumptions and Limitations

- CGModel models inhalation effects only[4,5]

[4] Phosgene "does not cause lung effects when absorbed through the skin, injected, or orally ingested" [26]
[5] "Inhalation is the most important route of exposure for phosgene" [18, p.16].

Table 11: Probability of lethality probit model parameters.

Parameter	Value	Reference
LCt_{50}	1500 mg·min/m^3 (10 min exposure duration, 15 L MV)	[22]
PS (probit slope)	10.5 ($\log_{10}$)[a]	[5, p.22]

[a] CGModel uses base 10 logarithms in its lethality probit model; thus, the argument of Φ changes by PS when D_{RE} changes by a factor of 10 (see Eq. 3) [5, p.3].

6 Appendix A: Table 7

The following methodology was used to develop Table 7, which describes the severity vector's dependence on effective dosage and effective time since exposure.

First, we performed a literature search to study the effects[6] of phosgene exposure (our findings are summarized in Sec. 7). Using the data we gathered, we then defined four concentration bands that (roughly) correspond to four increasingly severe levels of exposure. Table 12 describes these bands and summarizes our rationale for choosing them. These concentration bands correspond to the dosage bands in Table 7.

Next, for each S/S category in Table 1, we defined time-dependent severity values associated with the following four effective dosage values: $G_1 = \sqrt{4.1 \cdot 81}$, $G_2 = \sqrt{81 \cdot 182}$, $G_3 = \sqrt{182 \cdot 1215}$, and $G_4 = \sqrt{1215 \cdot 2025}$ mg·min/m^3 (these four values represent the dosage ranges shown in Table 7 and are used in the interpolation algorithm mentioned in Sec. 1). Graphs of these functions are given in Fig. 7. We defined these severity functions based on data from our literature search (summarized by Tables 13–31), the severity level definitions given in Table 1, and the notion that, for any fixed time t, severity should be an increasing function of dosage.

Finally, we combined the severity functions into the time dependent severity vectors shown in Table 7. That is, Table 7 is an alternate representation of the data shown in Fig. 7 (the dosage ranges in Table 7 actually refer to the discrete dosage values G_i that appear in Fig. 7).

7 Appendix B: Summary of Literature Search

Tables 13–31 summarize the data that was used to construct the severity functions in Fig. 7. Our literature search focused on collecting four pieces of information about phosgene exposures: the magnitude of the exposure (described in the tables below by concentration, exposure duration, dosage, and D_V[7]), the effects of the exposure (Effects/Comments), the time[8] at which the effects begin (T_{begin}), and the time at which the effects end (T_{end}). Usually

[6] Skin damage, systemic damage, respiratory damage, and ocular damage (see Table 1).

[7] Concentration, exposure duration, and/or dosage were given by the literature; we calculated D_V using the algorithm described in Sec. 1.

[8] Elapsed time after the beginning of the exposure.

Table 12: Concentration ranges that define (for 10–minute exposures) the dosage bands in Table 7.

Conc. Range (10 min. exposure)	Effects/Comments
0.1 - 2 ppm 0.41 - 8.1 mg/m^3	0.1 ppm: NIOSH REL (Recommended Exposure Limit), TWA [16][a] 0.1 ppm: OSHA PEL (Permissible Exposure Limit), TWA [16][b] 0.2 ppm: NIOSH REL (Recommended Exposure Limit), C (15 minute) [16][c] 0.6 ppm: AEGL 2 for 10 minute exposure [18, p.18]
2 - 4.5 ppm 8.1 - 18.2 mg/m^3	3 ppm: "irritation of mucous membrane surfaces of the eyes, nose, throat, and bronchi." [21, p.473] (1.9 ppm $\lesssim D_V/10 \lesssim$ 4.8 ppm)[d] 30 ppm·min: "beginning lung damage" [21, p.472] 3.6 ppm: AEGL 3 for 10 minute exposure [18, p.18] 2–5 ppm: "will induce mild respiratory symptoms, with prolonged exposure to such levels considered dangerous" [10] 3–5 ppm: "immediate irritant effects such as conjunctivitis, rhinitis, pharyngitis, bronchitis, lacrimation, blepharospasm conjunctival hyperemia, and upper respiratory tract irritation" [19] (cited in [28])
4.5 - 30 ppm 18.2 - 121.5 mg/m^3	(4 ppm $\lesssim D_V/10 \lesssim$ 8 ppm)[e] 50 ppm·min: "can result in delayed pulmonary edema" [19] (cited in [28]) ($D_V/10 \approx$ 4.8 ppm) 2 ppm: IDLH [16][f] ($D_V/10 \approx$ 5.5 ppm) "20 ppm by volume cause lung injuries in 2 min" [4] (cited in [28]) (11.5 ppm $\lesssim D_V/10 \lesssim$ 24 ppm)[g] 150 ppm·min: "clinical pulmonary edema" [21, p.472]; "will probably result in pulmonary edema" [19] (cited in [28])
30 - 50 ppm 121.5 - 202.5 mg/m^3	37 ppm: LC_{50} (1500 mg·min/m^3: LCt_{50} for 10 min exposure duration, 15 L minute volume) [22, p.II 11] "Exposure to levels greater than 50 ppm are considered massive and will likely be rapidly fatal" [10]

[a] TWA: "time–weighted average concentration for up to a 10–hour workday during a 40 hour workweek"

[b] "TWA concentrations for OSHA PELs must not be exceeded during any 8 hour workshift of a 40 hour workweek" [15]

[c] C: "ceiling REL .. unless noted otherwise, the ceiling value should not be exceeded at any time" [15]

[d] Exposure time between 1 and 30 minutes (see Fig. 4).

[e] Exposure time between 1 and 50 minutes (see Fig. 4).

[f] "As a safety margin, IDLH values are based on effects that might occur as a consequence of a 30–minute exposure" [15]

[g] Exposure time between 1 and 150 minutes (see Fig. 4).

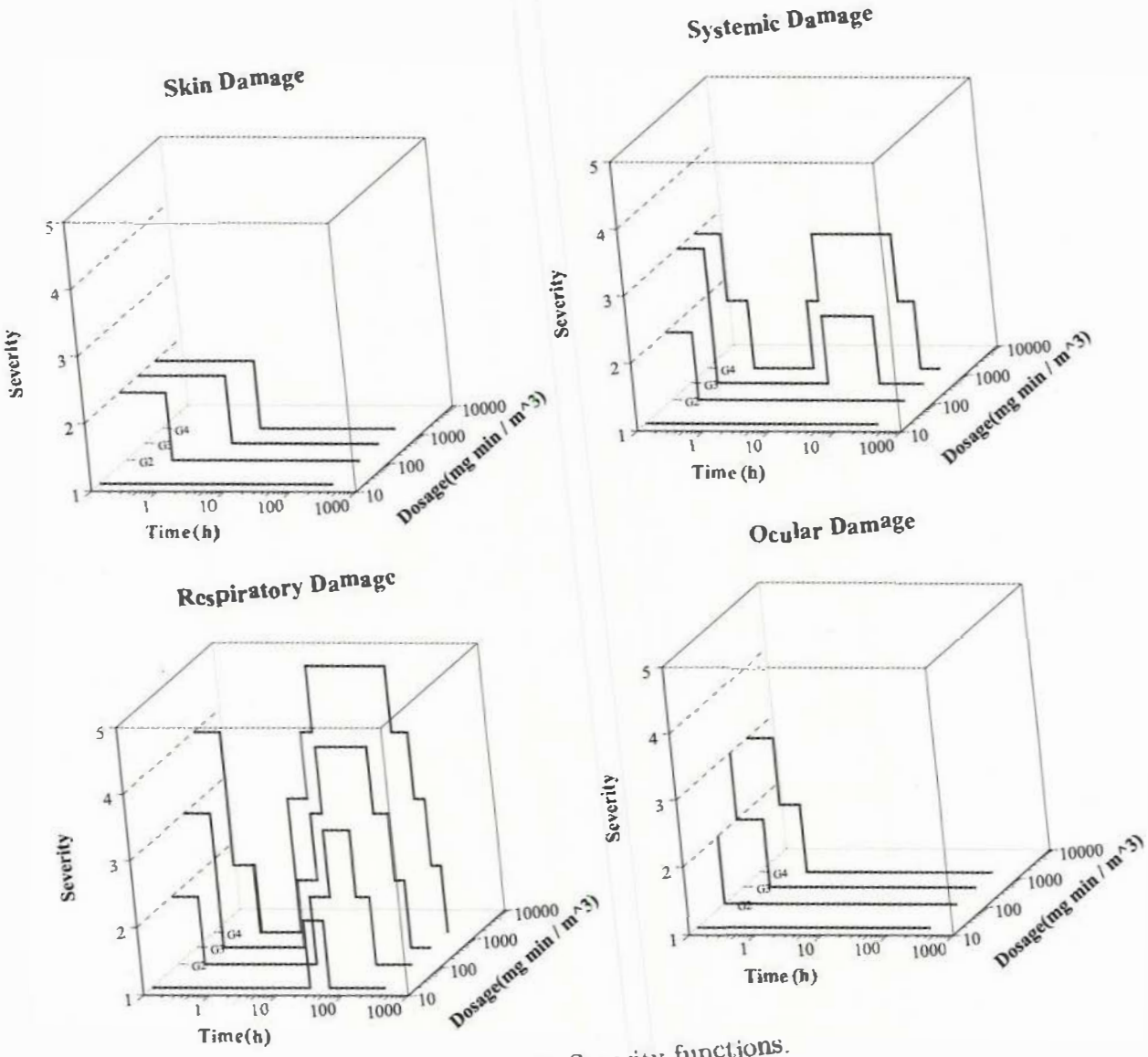

Figure 7: Severity functions.

only a subset of this information was available, however, and therefore many of the entries in Tables 13–31 are empty (indicated by "—"). Moreover, in many cases this information was given only in qualitative or semi quantitative terms. Tables 13–31 thus provide only general guidance for constructing the severity functions shown in Fig. 7. (Note: in Tables 13–31, unless otherwise indicated within the body of the table, units are as stated in the top row of each table).

To provide an overview of phosgene toxicity, we provide the following two excerpts:

> Human exposure in both the general population and occupational setting is primarily by inhalation. ... The primary route of exposure is by inhalation, the gas penetrates into the tissues of the respiratory tract and so only minimal amounts of phosgene are distributed in the body. The very short half-life (0.026 seconds) in aqueous solutions precludes a significant retention of phosgene in the body. ... The hydrolytic products of phosgene, i.e., hydrochloric acid and carbon dioxide, are disposed of by the body through normal physiological processes. Phosgene exerts its toxicity through acylation of proteins, as well as through the production of hydrochloric acid. The amino, hydroxyl and sulfhydryl groups in the protein appear to be the target for acylation leading to marked inhibition of several enzymes related to energy metabolism and a breakdown of the blood:air barrier. In all species studied, the lung is the major target organ. ... In all species the characteristic pathological feature is the delayed clinical manifestation of pulmonary edema, which is dose-dependent. Pathological changes in the terminal bronchioles and alveoli at low concentrations are typical of a pulmonary irritant, whereas at higher exposures pulmonary edema occurs, leading to interference with gas exchange and death. ... Phosgene exposure can result in eye and skin irritation. ... The target organ in humans, as in experimental animals, is the lung. After exposure to phosgene levels between 120 and 1200 mg/cu m-min, three distinct clinical clinicopathological phases have been reported. The initial phase consists of pain in the eyes and throat and tightness in the chest, often with shortness of breath, wheezing, and coughing; hypotension, bradycardia and rarely sinus arrhythmias can occur. The second or latent phase, which is often asymptomatic, can last as long as 24 hr depending upon the level and duration of exposure. In the third phase, pulmonary edema may develop, leading to death in some cases. Populations exposed to phosgene after industrial accidents have reported a wide variety of symptoms, including headache, nausea, cough, dyspnea, fatigue, pharyngeal pain, chest tightness and pain, intense pain in the eye and severe lacrimation. ... In view of the lack of exposure data ... the conclusions regarding the chronic effects of phosgene that can be drawn are limited. [11] (as cited in [28])

Although there is a paucity of acute human data containing known exposure concentrations and times, reports of human phosgene poisonings present a relatively consistent set of clinical effects and sequelae. After acute phosgene exposure, brief ($\leq$ 20 min) ocular and throat irritation, cough, nausea and vomiting, and dizziness are experienced, followed by a period ($\leq$ 24 h) of apparent well-being. After this clinical latency phase, cough accompanied by expectoration, a sensation of pain or tightness of the chest, shortness of breath, and a choking sensation are experienced. Clinical findings may include hemoconcentration, leukocytosis, rales, and pulmonary edema. After recovery, rapid shallow breathing, shortness of breath on exertion, and a sense of decreased physical fitness may persist for months. Pulmonary emphysema may occur with repeated exposure to phosgene. Epidemiology studies have shown no increase in cancer in workers exposed to phosgene compared with controls. No information concerning reproductive and developmental toxicity or genotoxicity was available [18, p.27].

Table 13: Data from literature search: cough.

Cough

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[11] (cited in [28])	—	—	120 – 1200	—	"initial phase"	—	"often with ... coughing"
[18, p.20]	"at least" 20 – 40	"5 to 10 seconds"	$\geq$ 1.7 – 6.7	?	immediate	—	accident (victim: "23-y-old man (healthy nonsmoker)")
[18, p.22]	19.4	—	—	—	—	—	
[18, p.22]		"massive" exposure			immediate	$\leq$ 5 minutes	accident (victim: "40-y-old male")
[26]	—	—	—	—	2 – 24	—	"As the damage progresses, the dyspnea will become more severe, and soon a cough will develop. If the damage is severe, the casualty will start coughing up a clear, foamy sputum, the plasma from his blood that has leaked into his alveoli."
[18, p.22]		"massive" exposure			$\approx$ 2	$\leq$ 5 days	"hacking cough"; accident (victim: "40-y-old male")
[7, p.38]	—	—	—	—	$\approx$ 1 – 24	—	"During the subsequent 'edema phase', the patient experienced progressive respiratory distress, sometimes symptoms like coughing ... returned"
[20, p.150]	> 12	—	—	—	—	—	"dry cough ... usually indicate[s] exposure to concentrations exceeding 3 ppm"

Table 14: Data from literature search: eye irritation.

Eye Irritation

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[21, p.473]	> 12	—	—	—	—	—	"initial irritation of mucous membrane surfaces of the eyes"
[11] (cited in [28])	—	—	120 – 1200	—	"initial phase"	—	"pain in the eyes"
[26]	—	—	—	—	"very shortly after exposure"	—	"casualty may notice irritation of the eyes"
[18, p.22]	16.2	—	—	—	—	—	
[18, p.22]	40.5	—	—	—	—	—	"severe eye irritation"
[18, p.22]		"massive exposure"			immediate	≤ 5 minutes	accident (victim: "40-y-old male")

Table 15: Data from literature search: nose irritation.

Nose Irritation

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[21, p.473]	> 12	—	—	—	—	—	"initial irritation of mucous membrane surfaces of the ... nose"
[26]	—	—	—	—	"very shortly after exposure"	—	"casualty may notice irritation of the ... nose"

Table 16: Data from literature search: throat irritation.

Throat Irritation

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[21, p.473]	> 12	—	—	—	—	—	"initial irritation of mucous membrane surfaces of the ... throat"
[11] (cited in [28])	—	—	120 – 1200	—	"initial phase"	—	"pain in the ... throat"
[26]	—	—	—	—	"very shortly after exposure"	—	"casualty may notice irritation of the ... throat"
[18, p.22]	12.6	—	—	—	—	—	
[7, p.38]	—	—	—	—	$\approx 1 - 24$	—	"During the subsequent 'edema phase', the patient experienced progressive respiratory distress, sometimes symptoms like ... burning sensation of the upper airways ... returned"

Table 17: Data from literature search: skin irritation.

Skin Irritation

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[10]		"massive exposures"			—	—	"possibility of dermal burning"
[10]		"lower exposures"			—	—	"some dermal irritation"
[1, p.6]	—	—	—	—	—	—	"If the skin is wet or moist, contact with phosgene vapor can cause irritation and redness of the skin"

Table 18: Data from literature search: chest tightness.

Chest Tightness

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[11] (cited in [28])	—	—	120 – 1200	—	"initial phase"	—	
[18, p.20]	"at least" 20 – 40	"5 to 10 seconds"	$\geq$ 1.7 – 6.7	?	$\leq$ 0.5	—	accident (victim: "23-y-old man (healthy nonsmoker)")
[7, p.38]	—	—	—	—	$\approx$ 1 – 24	—	"During the subsequent 'edema phase', the patient experienced progressive respiratory distress, sometimes symptoms like ... thoracic pain, chest tightness ... returned"
[20, p.150]	> 12	—	—	—	—	—	"chest tightness usually indicate[s] exposure to concentrations exceeding 3 ppm"

Table 19: Data from literature search: nausea and vomiting.

Nausea and Vomiting

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[18, p.27]		"acute exposure"			—	≤ 20 minutes	
[7, p.38]	—	—	—	—	$\approx 1 - 24$	—	"During the subsequent 'edema phase', the patient experienced progressive respiratory distress, sometimes symptoms like ... emesis and nausea ... returned"

Table 20: Data from literature search: latent period.

Latent Period

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[11] (cited in [28])	—	—	120 – 1200	—	"second phase"	≤ 24	"often asymptomatic"; length of latent phase "depend[s] upon the level and duration of exposure"
[18, p.16]	—	—	—	—	—	≤ 24	"clinical latency period"
[26]	—	—	—	—	"during or immediately after exposure"	—	"more commonly, there may be no effects during or immediately after exposure"
[26]	—	—	—	—	"hours later"	—	"the major effects ... do not occur until hours later"
[18, p.22]		"massive" exposure			≈ 5 minutes	≈ 2	accident (victim: "40-y-old male")
[7, p.38]	—	—	—	—	—	1 – 24	
[10]	—	—	—	—	—	0.5 – 24	"The patient is generally asymptomatic for 30 minutes to 24 hours after exposure."
[1, p.6]	—	—	—	—	"symptoms ... may cease when the patient is removed from exposure"	0.5 – 48	"asymptomatic interval of 30 minutes to 48 hours"

Table 21: Data from literature search: latent period.

Latent Period

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[19] (cited in [28]))	—	—	—	—	—	24 – 72	"Signs and symptoms of toxicity may be delayed, although rare, for 24 to 72 hours ... delayed effects usually occur within 24 hrs of exposure"
[8, p.6]	—	—	—	—	"the second phase, which may last for several hours postexposure"		"clinical signs and symptoms are generally lacking ... the length of this phase varies inversely with the inhaled dose"
[20, p.150]	"small doses", but greater than 120 $mg \cdot min/m^3$				—	24 - 48	"At dosages exceeding 30 $ppm \cdot min$, the initial irritation and respiratory symptoms are followed by a second (possibly asymptomatic) phase, the duration of which is inversely proportional to the inhaled dose. ... after small doses, 24 - 48 hours"
[20, p.150]	"large doses" greater than 120 $mg \cdot min/m^3$				—	1 – 4	"At dosages exceeding 30 $ppm \cdot min$, the initial irritation and respiratory symptoms are followed by a second (possibly asymptomatic) phase, the duration of which is inversely proportional to the inhaled dose. After large doses, it may be 1 - 4 hours"

Table 22: Data from literature search: pulmonary edema.

Pulmonary Edema

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[21, p.460]	—	—	—	—	1 – 24	—	"may develop into fulminating and life-threatening pulmonary edema"
[21, p.472]	—	—	> 600	—	—	—	"clinical pulmonary edema"
[11] (cited in [28])	—	—	120 – 1200	—	≤ 24 ("third phase")	—	"pulmonary edema may develop"
[27]	"high concentrations"				2 – 6	—	
[27]	—	—	—	—	≤ 48	—	"coughing up white to pink-tinged fluid (a sign of pulmonary edema)"
[11, p.39] (cited in [28])	"exposure without initial symptoms of eye or nose irritation"				≤ 48	—	—
[18, p.20]	"at least" 20 – 40	"5 to 10 seconds"	≥ 1.7 – 6.7	?	≤ 4	—	accident (victim: "23-y-old man (healthy nonsmoker)")
[18, p.22]	"massive" exposure				≤ 8	≤ 5 days	"severe pulmonary edema"; accident (victim: "40-y-old male")
[19] (cited in [28]))	—	—	202.5	—	—	—	"may cause pulmonary edema"
[19] (cited in [28]))	—	—	607.5	—	—	—	"will probably cause pulmonary edema"

Table 23: Data from literature search: pulmonary edema.

Pulmonary Edema

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[8, p.7]		"acute exposure"			< 24	3 – 5 days	"In general, this phase peaks approximately 24 hours after an acute exposure and, assuming lethality does not occur, recedes over the next 3 to 5 days. "
[20, p.150]	—	—	> 120	—	"third phase"	"within a few days"	"If the patient survives, clinical and radiological oedema resolve within a few days"
[20, p.150]	8.1	80	648	516	12 – 16	—	"Exposures to 2 ppm for 80 minutes will not cause any irritation but result in pulmonary oedema some 12 - 16 hours later"

Table 24: Data from literature search: dyspnea.

Dyspnea

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[26]	—	—	—	—	2 – 24	—	"Initially, this may be mild, and the eventual severity of the shortness of breath (dyspnea) will depend on the amount of exposure. As the damage progresses, the dyspnea will become more severe, and soon a cough will develop."
[26]		"very mild exposure"			6 – 24	—	
[26]		"severe exposure"			"within several hours"	—	
[26]		"severe exposure"			4 – 6	—	"[casualty] will find it increasingly difficult to breathe, even at rest"
[26]		"the average casualty"			6 – 8	—	"[casualty] ... may progress to have dyspnea while at rest"
[27]	—	—	—	—	≤ 48	—	"difficulty breathing"
[18, p.20]	"at least" 20 – 40	"5 to 10 seconds"	≥ 1.7 – 6.7	?	≤ 0.5	—	accident (victim: "23-y-old man (healthy nonsmoker)")
[18, p.22]		"massive" exposure			3	≤ 5 days	"mild dyspnea"; accident (victim: "40-y-old male")

Table 25: Data from literature search: dyspnea.

Dyspnea

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[18, p.22]		"massive" exposure			6	$\leq$ 5 days	"severe dyspnea and moist rales"; accident (victim: "40-y-old male")
[17, p.31]	—	30	—	—	$\approx$ 2	—	accident (victim: 55-year-old male); "presumed to have been exposed to phosgene released by chipping of brick"
[17, p.34]		"massive" exposure			3	—	
[20, p.150]	—	—	> 120	—	—	"several months"	"exertional dyspnoea ... may persist for several months"
[23, p.II-4]	—	—	—	—	—	—	"onset of dyspnea (shortness of breath) within four hours of exposure is usually a grave prognostic indicator"

Table 26: Data from literature search: potentiation with physical exertion.

Potentiation with Physical Exertion

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[26]		"very mild exposure"			6 – 24	—	dyspnea: "notice[d] ... first after heavy exertion; however, later [casualty] will become short of breath after any activity"
[26]	—	—	—	—	—	—	"Complete rest is extremely important. There were instances in World War I in which phosgene casualties who were breathing comfortably at rest collapsed and died after walking a few yards. Even a little exertion can greatly intensify the effects of these agents."
[9]	—	—	100	—	—	24 –72	"After exposure by inhalation, physical exertion should be avoided and strict bed rest enforced for between 24 and 72 h, particularly if the exposure dose was unknown or above 100 mg/m3-min (25 ppm-min)."
[20, p.150]	—	—	> 120	—	—	"several months"	dyspnea: "exertional dyspnoea ... may persist for several months"

Table 27: Data from literature search: potentiation with physical exertion.

Potentiation with Physical Exertion

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[23, p.II–3]	—	—	—	—	—	—	"This dyspnea usually occurs only after an hours–long clinically asymptomatic period that is inversely proportional to dose, and it can be brought on earlier by exertion."
[23, p.II–3]	—	—	—	—	—	—	"Management includes enforced rest (exertion leads to earlier appearance of effects and more severe effects), ..."
[23, p.II–5]	—	—	—	—	—	—	"Exertion after exposure will worsen the prognosis"

Table 28: Data from literature search: cardiovascular effects.

Cardiovascular Effects

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[11] (cited in [28])	—	—	120 – 1200	—	"initial phase"	—	"hypotension, bradycardia and rarely sinus arrhythmias can occur"
[27]	—	—	—	—	≤ 48	—	"low blood pressure, heart failure"
[18, p.20]	"at least" 20 – 40	"5 to 10 seconds"	$\geq 1.7 - 6.7$	?	≤ 4	—	hypotension, tachycardia; accident (victim: "23-y-old man (healthy nonsmoker)")

Table 29: Data from literature search: general respiratory symptoms.

Respiratory Systems (General)

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[21, p.473]	> 12	—	—	—	—	—	"initial irritation of mucous membrane surfaces of the ... bronchi"
[21, p.473]	> 80	—	—	—	—	—	"may produce apnea of several seconds duration, bronchoconstriction, bronchial epithelium desquamation, and inflammation of the bronchi"
[21, p.472]	—	—	> 120	—	—	—	"beginning lung damage"
[11] (cited in [28])	—	—	120 – 1200	—	"initial phase"	—	"often with shortness of breath, wheezing"
[18, p.20]	"at least" 20 – 40	"5 to 10 seconds"	$\geq 1.7 - 6.7$	?	≤ 4	—	tachypnea; accident (victim: "23-y-old man (healthy nonsmoker)")
[18, p.22]	40.5	—	—	—	—	—	"severe airway irritation"
[4, p.596] (cited in [28])	81	2	162	222	—	—	"cause[s] lung injuries"

Table 30: Data from literature search: fatigue/weakness.

Fatigue/Weakness

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[17, p.28]	—	—	—	—	2 hours – 3 days	"weeks or months"	based on observations of cases of phosgene exposure during World War I

Table 31: Data from literature search: miscellaneous.

Miscellaneous

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[21, p.462]	244.3	10	2443	2443	6	—	SWINE ("pulmonary architecture in swine is very similar to that of adult humans"): "swine show a marked decrease in pH, PO_2, O_2 saturation, dynamic compliance, and increased lung wet weight"
[7, p.38]	—	—	—	—	—	1 – 3 weeks	"Recovery normally was achieved within 1 to 3 weeks" (pulmonary edema, respiratory insufficiency, etc.)
[10]	—	—	—	—	—	weeks – years	"Documented cases show return to normal limits of lung function within a period of weeks, however complete recovery may take a period of years"
[1, p.5]	"low concentrations"	—	—	—	—	"symptoms ... may cease when the patient is removed from exposure"	"may cause no signs or symptoms initially, or symptoms may be due only to mild irritation of the airways"

Table 32: Data from literature search: miscellaneous.

Miscellaneous

Ref	Conc (mg/m^3)	Duration (min)	Dosage ($mg \cdot min/m^3$)	D_V ($mg \cdot min/m^3$)	T_{begin} (hours)	T_{end} (hours)	Effects/Comments
[1, p.16]	—	—	—	—	—	—	"Patients who survive for 48 hours usually recover"
[23, p.II–4]	—	—	—	—	—	—	"In most fatal cases, pulmonary edema reaches a maximum in 12 hours, followed by death in 24 to 48 hours. If the victim survives, resolution commences within 48 hours, and in the absence of complicating infection, there may be little or no residual damage."

8 Definitions, Acronyms, and Abbreviations

AEGL Acute Exposure Guideline Level.

Dosage Duration A measure of the effective duration of an exposure (this value is calculated elsewhere and passed to CGModel).

Effective Time Since Exposure (τ) Time elapsed since $ExposureDuration = DosageDuration/2$, that is, the time that has elapsed since the effective mid-point of the exposure. It is an analog of postexposure time for exposures of negligible duration.

Exposure Duration Time elapsed since the beginning of an exposure. This quantity, defined elsewhere and passed to CGModel, continues to increase even after the cessation of exposure. It should not be confused with dosage duration, which does not increase after the cessation of exposure.

Litter Time (τ_{litter}) First effective time since exposure at which $P = 0.25$. The exposed person is presumed to require assistance to reach medical care after this time.

MEG Military Exposure Guideline

Onset Time (τ_{onset}). First effective time since exposure at which $P = 0.995$. Indicates the time of first occurrence of signs or symptoms of injury.

PPM Parts per million (e.g., of phosgene gas) in air by volume.

RTD Return to Duty.

Seek Treatment Time (τ_{seek}) First effective time since exposure at which $P = 0.75$. The time at which an individual seeks treatment for his or her symptoms.

SME Subject Matter Expert.

S/S Sign/Symptom.

Time to Death Equal to 1.5 days if, and only if, the exposure is lethal.

Time to RTD (τ_{RTD}) If the exposure is nonlethal, the minimum performance is less than or equal to 0.75, and τ^* equals the time that elapses between the point at which $ExposureDuration = DosageDuration/2$ and the point at which P returns to 0.75, $\tau_{RTD} = \max\{\tau^*, \tau_{seek} + 24 \text{ hours}\}$. This RTD time is different than that used in AMedP-8, where it was assumed that no treatment was available. In the context of the software that uses CGModel, the lower bound ($\tau_{seek} + 24$ hours) on τ_{RTD} models time in the medical care system. Time to RTD is not defined if the minimum performance is greater than 0.75.

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